10.1111/j.1469-0691.2004.00971.x

Absence of carriage of glycopeptide-resistant enterococci by at-risk hospitalised patients in Malta

In a recent article in *Clinical Microbiology and Infection*, Cordeiro *et al.* [1] described the inter-hospital dissemination of glycopeptide-resistant enterococci (GRE) in Brazil. These organisms are isolated mainly from patients in intensive care, transplant and oncology units, and this has been attributed particularly to the use of combination antimicrobial therapy involving vancomycin and anti-anaerobe agents, coupled with prolonged hospitalisation [2,3]. GRE have a major impact on the management of patients and wards [4], and gastrointestinal colonisation with GRE is difficult to eradicate once it is established [5,6].

There have been no previous reports of GRE in Malta, which is an island in the Mediterranean with a population of 450 000. This contrasts with other countries in the region, where the prevalence of GRE in blood culture isolates has been reported to reach 2% [7]. Glycopeptide use (predominantly teicoplanin) in Malta is common because of the high prevalence of methicillin-resistant *Staphylococcus aureus* infections in St Luke's Hospital, which is the only tertiary care hospital (with 900 beds, including a 13-bed intensive care unit) on the island. In order to establish whether the at-risk population in this hospital harboured GRE, a 4-month screening programme, concentrating on the intensive care unit, was performed in June to September 2002.

In total, 310 rectal swabs were taken from 300 patients and inoculated into VRE broth (Oxoid, Basingstoke, UK) supplemented with meropenem (Oxoid) 1 mg/mL and colistin sulphate (Pharmax, Bexley, UK) 7.5 mg/L (to suppress the growth of meropenem-resistant *Pseudomonas aeruginosa*). Control experiments demonstrated that this broth supported the growth of the standard strains *Enterococcus faecalis* NCTC 12201 (*vanA*$^+$), *E. faecalis* ATCC 33186 (glycopeptide-susceptible) and *Enterococcus faecium* ATCC 51559 (*vanA*$^+$). Following incubation at 37°C for 16 h, the broths were streaked on to VRE agar (Oxoid) supplemented with meropenem 0.5 mg/L and vancomycin (Oxoid) 6 mg/L. Following incubation at 37°C for 24–48 h, putative enterococci were identified to the species level with the API STREP system (bioMérieux, Marcy l'Etoile, France), and susceptibilities to vancomycin and teicoplanin were determined with Etests (AB Biodisk, Solna, Sweden).

Only one isolate of GRE was recovered during the 4-month screening period. This isolate was identified as *E. faecium* (vancomycin MIC >32 mg/L; teicoplanin MIC 32 mg/L) and was shown by PCR to carry the *vanA* gene. The patient was a poultry farm worker and was screened on the first day following admission to the intensive care unit. The patient was not receiving any antimicrobial therapy and had no previous history of prolonged hospitalisation. Interestingly, the presence of GRE in Europe has been linked to the use of avoparcin in animal husbandry [8]. Avoparcin was used in poultry farms in Malta until August 1999, when its importation was banned, but residual stocks may well remain at some farms.

GRE are now found worldwide, but these results confirmed that the prevalence of GRE within St Luke's Hospital remains negligible. Since St Luke's is the only tertiary hospital on the island, it seems that the prevalence of GRE in Malta is considerably lower than that reported in many other European countries.

ACKNOWLEDGEMENTS

We gratefully acknowledge the assistance of the staff of the Infection Control and Health Information Units, and financial support from the Malta Health Division.

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10.1111/j.1469-0691.2004.00945.x

Zygomycosis in neutropenic patients with past *Aspergillus* infection: a role for posaconazole?

In the March 2004 supplement of *Clinical Microbiology and Infection*, Donnelly and De Pauw [1] published a review on voriconazole, with emphasis on its use for treating cases of candidosis, invasive aspergillosis, and some more uncommon mycoses caused by *Scedosporium* spp. and *Fusarium* spp. Voriconazole has little activity against *Sporothrix schenckii* and zygomycetes such as *Mucor* spp., *Rhizopus* spp. and *Absidia* spp. [1]. Thus, selective pressure from prolonged courses of voriconazole, or increased survival among profoundly immunosuppressed patients, may explain the higher incidence of zygomycosis in patients who remain at obvious risk for invasive fungal infections [2].

Marty *et al.* [2] have described breakthrough zygomycoses in recipients of allogenic haematopoietic stem-cell transplants since the introduction of voriconazole, and we would like to report a similar case of a man aged 65 years, diagnosed with acute myeloid leukaemia, who received voriconazole for proven neutropenia-induced invasive pulmonary aspergillosis (i.e., respiratory samples positive on direct examination and culture for *Aspergillus fumigatus*; serum galactomannan positive on several occasions; compatible thorax lesions visualised by computerised tomography scan). While receiving voriconazole, the patient developed a massive maxillary and ethmoidal sinusitis, with proptosis of the right eye showing hyphal elements on direct examination of tissue samples. Culture remained sterile. Therapy was altered to amphotericin B, but persistence of the lesions prompted a Caldwell–Luc debridement. Tissue samples now revealed fungal elements, seen typically in cases of zygomycosis, comprising thick-walled, aseptate right-angle branching filaments (Fig. 1) [3]. Culture yielded no growth. The patient's therapy was changed to oral posaconazole 800 mg daily [4,5]. The patient recovered and continues to receive posaconazole 400 mg daily without major side effects.

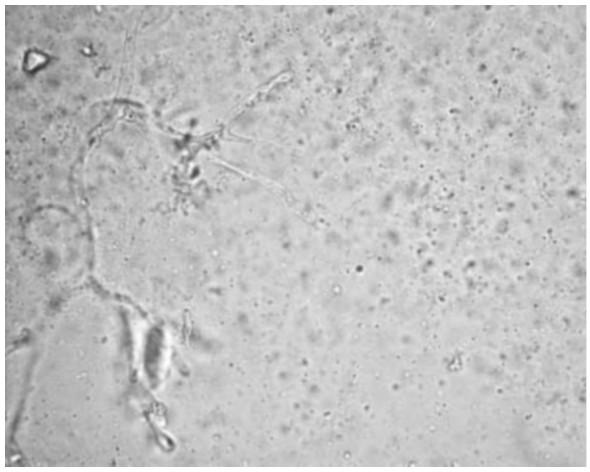

Fig. 1. Thick-walled, aseptate right-angle branching filaments, seen typically in cases of zygomycosis

We conclude that posaconazole is a potentially useful therapy for patients developing breakthrough zygomycosis after long-term use of voriconazole. A good response has already been reported in a case of invasive aspergillosis treated with posaconazole [6], but it is not yet clear whether this drug is appropriate for the first-line treatment of non-*Candida* mycoses in these patients.

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